cedure was done, however, the small amount of pituitary tissue in the otherwise empty sella had basophilic hyperplasia and no adenoma. In addition, partial pituitary hypofunction is suggested by plasma gonadotropin levels that were inappropriately low for her postmenopausal state. Thyroid function was normal. Several management issues arose during the course of this patient's illness that deserve comment. Selective venous catheterization would have been helpful in establishing the pituitary as the source of inappropriate ACTH production. In a case of pituitary-dependent Cushing's and an empty sella, it is probably still warranted to surgically explore the pituitary fossa for a possible adenoma. Bilateral adrenalectomy remains, however, a reasonable treatment alternative.

The question of whether this patient had Cushing's disease or Cushing's syndrome on another basis cannot ultimately be settled without a postmortem examination. The weight of the evidence suggests a pituitary basis. The factors supporting a primary pituitary aberration are the inappropriate rise in the ACTH level (though still within the normal range) in the presence of extreme hypercortisolism and its partial suppressibility. Other possible causes for Cushing's syndrome appear to be reasonably excluded. Documentation by CT scan of enlargement of both adrenal glands rules out a unilateral adrenocortical adenoma as the source of this patient's hypercortisolism. An ectopic source for ACTH is also unlikely for several reasons. The patient's long clinical course (six years), negative findings on a chest x-ray film, normal 24-hour urinary excretion of 5-hydroxyindoleacetic acid and partially suppressible hypercortisolism are inconsistent with an ectopic source for ACTH. Furthermore, the ACTH level in this paraneoplastic syndrome tends to be much higher than occurred in this patient. The failure of high-dose dexamethasone to suppress urinary 17-hydroxycorticosteroid excretion by at least 50% may argue against pituitary-dependent Cushing's disease, but 15% to 30% of these patients fail to suppress to this degree.[14]

This case also has potentially interesting implications concerning the natural history of Cushing's disease. If this patient's primary defect was at the level of the hypothalamus resulting in inappropriate stimulation of the pituitary, this would have caused development of a pituitary adenoma, ACTH hypersecretion and cushingoid features. Infarction of the adenoma may have then occurred, resulting in an empty sella. Of other possible causes of an empty sella, a developmentally incomplete sellar diaphragm cannot be completely excluded, whereas involution of a previously hypertrophied and overstimulated pituitary due to end-organ failure appears highly unlikely.

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Homonymous Hemianopia Due to Cerebral Air Embolism From Central Venous Catheters

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PERCUTANEOUS PLACEMENT of a central venous catheter has become a routine bedside procedure. Immediate mechanical complications include direct trauma to nerves, arteries and pleura and embolization of air or catheter fragments. Delayed complications are primarily infectious and thrombotic in nature.[1,2] The following reports of cases illustrate an unusual complication of central venous catheters—homonymous hemianopia due to cerebral air embolism—and highlight the need to implement precautions to prevent this complication.

Reports of Cases

CASE 1. The patient, a 60-year-old man, was admitted for control of malignant hypertension and unstable angina. Evaluation showed diffuse arteriosclerosis, which included carotid and renal artery disease as well as 70% occlusion of his left anterior descending and right coronary arteries. The patient underwent right carotid endarterectomy in April 1981 and coronary-artery-bypass graft operation three weeks later. Both procedures were uneventful. Five days after the cardiac operation the patient was transferred to the ward with a left subclavian venous catheter in place.

(Kearns PJ Jr, Haulk AA, McDonald TW: Homonymous hemianopia due to cerebral air embolism from central venous catheters. West J Med 1984 Apr; 140:615-617)

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Submitted, revised, June 22, 1983.

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Two hours later, the patient unwittingly disconnected the catheter tubing to walk to the bathroom. He was found standing in the doorway with the central venous catheter disconnected, complaining of shortness of breath and back, shoulder and jaw pain. On physical examination, the patient had a heart rate of 120 per minute, a respiratory rate of 30 and a blood pressure of 200/170 mm of mercury. No blood was noted to escape from the disconnected catheter. He had a left homonymous hemianopia and diminished sensation of the left side of his body; both findings were new. His supine central venous pressure measured 4 cm of water, which remained stable for the next 24 hours. The patient was placed at bed rest and given high-flow oxygen therapy. His blood pressure gradually fell to 130/80 mm of mercury during the first hour and his respirations returned to 20 per minute.

A diagnosis of air embolism from the disconnected catheter was considered. A computed tomogram (CT) of the head done 12 hours after the initial symptoms showed a circumscribed 4-mm air-density lesion (about −400 Hounsfield units) in the right calcarine cortex consistent with an air embolus in the right posterior cerebral artery (Figure 1). This finding correlated well with the patient's symptoms of left homonymous hemianopia and left-sided sensory abnormalities. The patient's sensory deficits totally resolved during the ensuing five days, and the visual field defect improved slightly. A repeat CT scan five days after the initial study showed complete resolution of the air embolus, with a residual area of low attenuation in the right occipital cortex consistent with cerebral infarction. Review of the findings of the physical examination, cardiac catheterization and echocardiogram showed no evidence of ventricular or atrial septal defect or a patent foramen ovale. He was discharged and readmitted eight months later for aortofemoral bypass with renal artery reconstruction. At that time he had only a residual inferior left quadrantanopia.

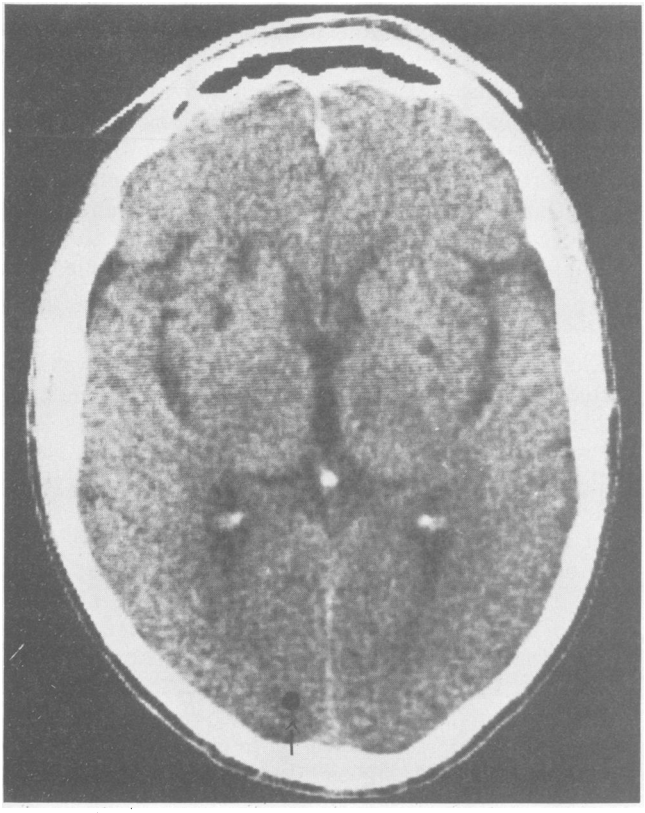

Figure 1.—Computed tomogram of the patient in case 1 obtained 12 hours after the onset of symptoms, showing an air-density lesion **(arrow).** A repeat scan 5 days later showed resolution of the air-density lesion with a residual area of low attenuation.

CASE 2. The patient, a 63-year-old man with diabetes mellitus and coronary artery disease, was admitted because of bacteremia, cellulitis and staphylococcal abscesses of the left lower extremity. The patient had poor peripheral venous access and required a central venous catheter for intravenous administration of antibiotic therapy. Two days after placement of the catheter, he inadvertently removed the catheter despite stabilization by a suture ligature. A sterile dressing was applied. Within an hour the patient noted decreased vision in his left eye, but he did not report this symptom at that time. A second central venous catheter was placed without incident, and antibiotic therapy was continued. Five days after placement of the second catheter, the patient complained of persistent left eye "blindness" and stated that the onset had occurred at the time of the dislodgement of the first central venous catheter. On physical examination a left homonymous hemianopia was found. A CT scan showed an area of low attenuation in the occipital cortical region consistent with cerebral infarction. Digital subtraction angiography showed no evidence of carotid artery disease. Neither echocardiogram nor cardiac catheterization showed evidence of a mural thrombus or valvular vegetation. A two-week course of antibiotics was completed and the infection resolved. The left homonymous hemianopia was unchanged when the patient was seen a year following the cerebral infarction. The CT scan has remained unchanged.

Discussion

The case of a patient who had a cerebral air embolus from a disconnected central venous catheter with a transient homonymous hemianopia was reported by Menkin and Schwartzman.[3] Postulated mechanisms for the neurologic symptoms following air embolism include obstruction of the outflow tract of the right ventricle with consequent decrease in cardiac output, intense cerebral vasospasm induced by air emboli passing though intracranial vessels and actual occlusion of cerebral vessels by trapped air emboli.[4,5]

The findings of the CT scan in the first patient may be the first clear clinicoanatomic correlation between neurologic symptoms and an air embolus in the brain. In this patient the underlying pathophysiologic feature was obstruction to cerebral blood flow by the air embolus, which caused ischemia and infarction. The timing of the CT scan showed that clinically significant air

TABLE 1.—*Accumulated Experience of Cerebral Air Emboli*

Study, Year	*Patients N**	*Catheter Associated N*	*Neurologic Symptoms (N)*	*Mortality %*
Green and Nemir, 1971[11]	1	1	**Blindness (1), paresis**	...
Hoshal and Fink, 1969[12]	2	2	Semicoma (1)	...
Ponsky, 1971[6]	1	1	**Coma (1), seizures**	100
Menkin and Schwartzman, 1973-1974[3]	5	1	Hemianopia (1)	40
Coppa et al, 1976-1980[13]	14	14	**Coma, seizure (9), hemiplegia**	30
Grace, 1977[14]	2	2	Hemiparesis (2), coma	...
Peters and Armstrong, 1978[5]	2	2	**Monoplegia (1)**	50
Michel et al, 1982[15]	1	1	Coma (1)	100
Eisenhauer et al, 1982[16]	3	3	**Seizure (2)**	...
TOTALS	31		19	

*Total number of patients in cited report who had presumed air emboli.

emboli can persist up to 12 hours and are resorbed within five days.

The mechanism by which venous air emboli are delivered to the systemic circulation is controversial. The previously postulated mechanisms presumed obstruction of the outflow tract of the pulmonary artery by air embolism with an increase in the pulmonary artery pressure transmitted to the right atrium. There is subsequent shunting of blood through an existing anatomic defect, such as a patent foramen ovale.[6] Alternative explanations must now be considered because the first patient had a low central venous pressure during the entire period of observation. In addition, neither an atrial septal defect nor a patent foramen ovale could be found. Intrapulmonary, physiologic right-to-left shunts have been found in dogs and in humans.[7] These are presumed to be patent and functional and are of sufficient size to allow passage of air emboli. Butler and Hills showed in dogs that aminophylline and vasodilators "open up" the pulmonary venous-arterial barrier and allow passage of air emboli to the arterial circulation.[8] The appearance of systemic air emboli with normal right-sided pressures in case 1 supports the significance of intrapulmonary shunts in the pathophysiology of the cerebral air embolus.

The neurologic symptoms of these two patients were assumed initially to be due to malignant hypertension, atherosclerotic disease or an idiopathic cause. Because of a high index of suspicion for cerebral air embolism, this diagnosis was confirmed in the first patient, and all other possible causes were excluded in the second patient. In all, 19 cases with the clinical diagnosis of cerebral air embolism due to central venous catheters have been reported, 15 within the past five years (Table 1). Because of the increasing use of central venous catheters for central pressure monitoring, for alimenting, in placing temporary pacemakers and in subclavian catheter dialysis, the incidence of air emboli with neurologic complications is likely to increase. Because these two patients were standing during their episodes, the air embolus followed a predictable route and traveled to the right posterior circulation. As noted by Ohkuda and co-workers,[9] venous air emboli usually do not cause symptoms, possibly because in supine patients the embolus can travel to neurologically silent areas. Because of this and the misconception that significant air emboli cause cardiovascular collapse, many air emboli may go unnoticed.

Because of the unknown incidence of this complication and the increasing use of the procedure, the following precautions are recommended for all central venous catheter placements:

- Follow proper insertion technique[10];
- Use Luer-lock connections to the catheter to avoid disconnection;
- Place suture-ligatures to prevent movement of the catheter, best accomplished with catheters equipped with ligature eyelets.

If these simple precautions are followed, cerebral air embolism should be reduced. When the possibility of air embolism exists, the use of computed tomography for the early diagnosis of cerebral air embolism should expedite the diagnosis and institution of definitive therapy.

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